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## PAPER

## Genetic architecture of verbal abilities in children and adolescents

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## Abstract

*The etiology of individual differences in general verbal ability, verbal learning and letter and category fluency were examined in two independent samples of 9- and 18-year-old twin pairs and their siblings. In both age groups, we observed strong familial resemblance for general verbal ability and moderate familial resemblance for verbal learning, letter and category fluency. All familial resemblance was explained by genetic factors. There was significant covariance among the tests, which was stronger in magnitude in the adolescent cohort. The covariance was mainly explained by genetic effects shared by subtests, both in middle childhood and in late adolescence. In addition to a shared set of genes that influenced all phenotypes, there were also genetic influences specific to the different verbal phenotypes.*

## Introduction

The development of verbal abilities forms a crucial part of a child's maturation process. Verbal abilities are key components for acquiring language, and learning how to read and write. Moreover, verbal abilities are needed for good social communicative functioning. A thorough understanding of the etiology of individual differences in verbal abilities, of how different verbal abilities are related to one another, and about the development of these abilities over time, is therefore highly relevant.

The development of the mental lexicon, which represents a mental repository of word-specific information, constitutes an important aspect of verbal abilities. Verbal learning and memory subserve the mental lexicon, by a process of registration, storage, retention, and retrieval of verbal information (Lezak, 1995). Tests such as the Rey's Auditory Verbal Learning Test (AVLT) or the California Verbal Learning Test (CVLT) yield reliable indices of declarative learning and memory (Lezak, 1995; Mulder, Dekker & Dekker, 1996; Van den Burg & Kingma, 1999) and can be used to study word learning processes.

Tasks of verbal fluency measure the spontaneous generation of words, and provide tests for language production and retrieval. Performance on these tests depends on size of and access to the lexicon (Mitrushina, Boone & D'Elia, 1999) and these tasks thus provide a proxy to study the output of the mental lexicon. Verbal

fluency tasks also require cognitive flexibility (for rapidly shifting from one word to the next) as well as response inhibition, and therefore also provide a test of executive functioning (Mitrushina *et al.*, 1999). Two forms of verbal fluency tests are the most common. Letter or phonological fluency evaluates the production of words starting with a certain letter. Semantic or category fluency measures the generation of words belonging to a certain semantic category. According to Levelt's model of speech production (Levelt, 1999, 2001), these two tasks require input from different levels of word production: letter fluency depends on lower-order phonological information, while category fluency requires higher-order semantic information processing. Both levels of word production show reliable individual differences (Bouma, Mulder & Lindeboom, 1996; Lezak, 1995). To what extent these individual differences overlap is unclear.

Verbal learning and fluency are both positively related to general verbal intelligence (Bishop, Knights & Stoddart, 1990; Bolla, Gray, Resnick, Galante & Kawas, 1998; Bouma *et al.*, 1996; Mulder *et al.*, 1996). Moreover, both tests are related to the mental lexicon and we thus expect them to be associated with each other. Little is known about the relation between these abilities over the course of development. Verbal learning and fluency continue to improve during late childhood (Sincoff & Sternberg, 1988; Van den Burg & Kingma, 1999) and adolescence (Clark, Paul, Williams, Arns,

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Fallahpour, Handmer & Gordon, 2006; Levin, Culhane, Hartmann, Evankovich, Mattson, Harward, Ringholz, Ewing-Cobbs & Fletcher, 1991). To what extent improved performance on these tests relates to the development of general verbal abilities is unclear.

More insight into the relationship between general verbal abilities and the more specific abilities verbal learning, letter and category fluency is therefore of interest to the fields of both developmental psychology and language. Moreover, impaired performance on tests of verbal learning and verbal fluency has been associated with a variety of psychiatric conditions, including schizophrenia (Appels, Sitskoorn, Westers, Lems & Kahn, 2003; Chen, Chen & Lieh-Mak, 2000; Egan, Goldberg, Gscheidle, Weirich, Rawlings, Hyde, Bigelow & Weinberger, 2001; Simon, Cattapan-Ludewig, Zmilacher, Arbach, Gruber, Dvorsky, Roth, Isler, Zimmer & Umbricht, 2007; Weickert, Goldberg, Gold, Bigelow, Egan & Weinberger, 2000), depression (Videbech, Ravnkilde, Kristensen, Egander, Clemmensen, Rasmussen, Gjedde, Rosenberg & Gade, 2003), attention deficit hyperactivity disorder (Geurts, Verte, Oosterlaan, Roeyers & Sergeant, 2004; Marzocchi, Oosterlaan, Zuddas, Cavolina, Geurts, Redigolo, Vio & Sergeant, 2008) and autism (Geurts *et al.*, 2004). To advance our knowledge about the etiology of these disorders, it may be of value to get an insight into the factors that influence variation in verbal learning and fluency and its overlap with general verbal ability.

The etiology of individual differences in cognitive abilities can be studied in samples of genetically related individuals. In the last two decades, twin, family and adoption studies have generated a wealth of knowledge about the genetic and environmental influences on various cognitive abilities, including verbal abilities. These studies suggest that genetic influences on general verbal intelligence become more important over the lifespan in explaining individual differences, while the influence of shared environmental factors (influences from the environment that are shared between family members, which make relatives more alike) decrease. Within the Twins Early Development Study (TEDS), variance in general verbal intelligence in infants was found to be strongly influenced by shared environmental effects, and only moderately (about 25%) by genetic effects (Price, Eley, Dale, Stevenson, Saudino & Plomin, 2000). Twin family studies from the Netherlands show that the heritability increases to about 50% in middle childhood (Hoekstra, Bartels & Boomsma, 2007), and to about 85% in adulthood (Hoekstra *et al.*, 2007; Posthuma, De Geus & Boomsma, 2001; Rijdsdijk, Vernon & Boomsma, 2002). In parallel, shared environmental influences decrease at later ages in childhood and become non-significant by adolescence. The Colorado Adoption Project (CAP) has collected data on the development of cognitive abilities in adopted children and their adoptive and biological parents, and in non-adoptive families. The heritability of verbal abilities

increased from 11% when the children were 4 years of age (Rice, Carey, Fulker & DeFries, 1989) to 24% and 26% when the children were 7 (Alarcón, Plomin, Corley & DeFries, 2003) and 12 years old (Alarcón *et al.*, 2003; Alarcón, Plomin, Fulker, Corley & DeFries, 1998). When the offspring were 16 years old, heritability was 64% (Alarcón, Plomin, Fulker, Corley & DeFries, 1999). Thus, both adoption and twin studies indicate that genetic factors become increasingly important for explaining variance in general verbal abilities when children grow older.

In a comprehensive review and meta-analysis, Stromswold (2001) examined the evidence for genetic effects on lexical, phonological, morphosyntactic and written language skills. The meta-analysis of three twin studies that examined vocabulary acquisition in late infancy (Dale, Dionne, Eley & Plomin, 2000; Ganger, Pinker, Chawla & Baker, 1999; Reznick, Corley & Robinson, 1997) included 1247 monozygotic (MZ) and 1152 dizygotic (DZ) twin pairs. Genetic factors accounted for 29% of the individual differences in vocabulary size, and shared environmental influences explained 66% of the variance, while nonshared environmental factors accounted for little of the variance (5%). The meta-analysis of five studies of vocabulary in 3- to 12-year-old children (Fischer, 1973; Foch & Plomin, 1980; Mather & Black, 1984; Segal, 1985; Thompson, Detterman & Plomin, 1991), including 330 MZ and 237 DZ twin pairs, found that genetic factors accounted for 53% of the variance, and shared and nonshared environmental influences explained respectively 18% and 29% (Stromswold, 2001). Two more recent studies investigated the heritability of vocabulary in 4.5-year-old twins and reported estimates of respectively 32% (Samuelsson, Byrne, Quain, Wadsworth, Corley, DeFries, Willcutt & Olson, 2005) and 52% (Kovas, Hayiou-Thomas, Oliver, Dale, Bishop & Plomin, 2005). Together, these studies suggest an increasing influence of genetic effects on vocabulary which is accompanied by decreasing shared environmental influences during development.

The genetic and environmental influences on early childhood verbal short-term memory and verbal fluency have been studied both in twins from TEDS, and in an international twin sample. In 4.5-year-old twins from TEDS, the heritability of both abilities was moderate (respectively 36% and 40%), and shared environmental influences were not significant (Kovas *et al.*, 2005). In a combined sample of Australian, Scandinavian, and American children (Samuelsson *et al.*, 2005), more prominent genetic (57%) and shared environmental influences (29%) were detected for verbal short-term memory. In a study of 6-year-old twin pairs with an overrepresentation of children at risk for language impairment, phonological short-term memory was found to be under substantial (61%) genetic influence, while shared environmental effects were non-significant (Bishop, Adams & Norbury, 2006).

Studies into specific verbal abilities in adult samples are scarce. Swan *et al.* (Swan, Reed, Jack, Miller, Markee, Wolf, DeCarli & Carmelli, 1999) examined verbal learning and memory in aging male twins, and reported a heritability of 56%. In the same study, individual differences in verbal fluency were explained by moderate genetic influences (34%), a (statistically non-significant) shared environmental component (18%) and nonshared environmental influences (48%; Swan & Carmelli, 2002). In 18- to 25-year-old female twins, free recall of unrelated words and categorized words were both moderately heritable (respectively 55% and 38%; Volk, McDermott, Roediger & Todd, 2006). Ando, Ono and Wright (2001) studied verbal and spatial working memory in a sample of 16- to 29-year-old twins, and also included verbal and spatial ability scores on a standardized intelligence test. Verbal working memory was moderately heritable (43–48%), while general verbal ability was under strong genetic influence (65%).

To summarize, twin and adoption studies into general verbal intelligence and vocabulary indicate increasing genetic and decreasing shared environmental influences over the course of development. Studies on verbal memory and verbal fluency in early childhood and in adulthood suggest moderate to strong genetic influences, while shared environmental effects do not appear to play a major role.

If different verbal abilities are influenced by common genetic factors, then we would expect that as the importance of genetic influences increases during development, the association among tests would also increase. We address this question through the use of multivariate genetic analyses of data on general verbal ability (as measured with the Wechsler verbal intelligence scale), verbal learning and letter and category fluency that were collected in two different birth cohorts. Data were collected in 9-year-old twins and their siblings (the 'child cohort'); and in 18-year-old adolescent twins and their siblings (the 'adolescent cohort'). The study includes siblings of twins so that it was possible to test whether the covariance structure in the data is the same for twins and siblings. Additionally, if there are no twin-sibling differences, the inclusion of siblings in the study greatly increases the statistical power to detect genetic and shared environmental effects (Posthuma & Boomsma, 2000).

## Method

### Participants

All twin families were recruited via the Netherlands Twin Register (NTR), kept by the Department of Biological Psychology at the VU University in Amsterdam (Bartels, Van Beijsterveldt, Derks, Stroet, Polderman, Hudziak & Boomsma, 2007; Boomsma, De

Geus, Vink, Stubbe, Distel, Hottenga, Posthuma, van Beijsterveldt, Hudziak, Bartels & Willemsen, 2006). The current project includes data from two longitudinal studies. The child cohort took part in a study into brain development and cognition in early puberty and consisted of 112 9-year-old twin pairs (mean age 9.10 years,  $SD = 0.10$ ) and their 9- to 14-year-old siblings ( $n = 100$ , mean age 11.84 years,  $SD = 1.16$ ). The brain development of these children was studied by structural brain imaging; cognitive development was assessed using an extensive neuropsychological test protocol. The current study focused on the results of the verbal tasks. Since these children took part in an MRI study, there were some exclusion criteria, such as having a pacemaker or braces (Van Leeuwen, Van den Berg & Boomsma, 2008). There were 23 monozygotic male (MZM) and 25 monozygotic female (MZF) twin pairs, 23 dizygotic male (DZF) and 21 dizygotic female (DZF) twin pairs, and 20 dizygotic twin pairs of opposite sex (DOS). For the same sex twin pairs, zygosity determination was based on DNA polymorphisms (90 twin pairs) or on longitudinally collected questionnaire items (two pairs; Rietveld, Van der Valk, Bongers, Stroet, Slagboom & Boomsma, 2000). There were 43 brothers and 57 sisters. The adolescent cohort took part in a longitudinal study into the development of cognition and behavioral problems (Hoekstra *et al.*, 2007; Bartels, Rietveld, Van Baal & Boomsma, 2002). This group consisted of 186 families of 18-year-old twin pairs (mean age 18.18 years,  $SD = 0.21$ ) and their siblings ( $n = 93$ , mean age = 18.51 years,  $SD = 4.73$ ). There were 33 MZM, 34 DZM, 44 MZF, 38 DZF, and 37 DOS pairs. The zygosity of the same sex twin pairs was determined by DNA or blood group polymorphisms (139 and nine pairs, respectively), or questionnaire items (one pair; Rietveld *et al.*, 2000). There were 46 male and 47 female siblings. Both studies were approved by the Central Committee on Research Involving Human Subjects and the institutional review board of the VU University Amsterdam. Written informed consent was obtained from participants who were 18 years of age or older, and from the parents of underaged participants.

### Test procedures

In both study cohorts, the cognitive testing took place at the laboratory of the VU University. The cognitive test protocol in the child cohort started in the morning and took approximately 5 hours to complete, including breaks. The families of the adolescent cohort were seen by a pediatrician in the morning, who studied their physical development. These twin families completed the cognitive test protocol in the afternoon, which took about 3.5 hours, including a break. In both studies, children from the same family were tested on the same day in different rooms by experienced test administrators.



### Measures

All participants in the child cohort, and the siblings from the older cohort who were younger than 16 years, completed the full Wechsler Intelligence Scale for Children-Third edition (WISC-III; Wechsler, Kort, Compaan, Bleichrodt, Resing, Schittekatte, Bosmans, Vermeir & Verhaeghe, 2002). Verbal IQ (VIQ) scores were determined as the standardized score on five verbal subtests. The standardized scores were based on results of same-aged children from the Netherlands. All participants of 16 years or older completed 11 subtests from the Wechsler Adult Intelligence Scale-Third edition (WAIS-III; Wechsler, 1997). Verbal IQ was calculated as the mean subtest score on six verbal subtests. The subtests were standardized for the appropriate age group, based on a population sample of same-aged subjects in the Netherlands.

In the child cohort, verbal learning and memory were assessed using the Dutch version of Rey's Auditory Verbal Learning Test (AVLT; Van den Burg & Kingma, 1999). In this task, a list of 15 unrelated, concrete nouns (e.g. bird, pencil) is presented over five learning trials, and immediate recall is tested following each presentation. Verbal learning was measured as the total number of correct words over the five learning trials. The test-retest reliability of the AVLT has been examined using parallel tests in 225 Dutch school children (Van den Burg & Kingma, 1999). Verbal learning was found to be the most reliable measure of the task, with a test-retest correlation of .70.

The 18-year-old twins and their siblings completed the Dutch adaptation of the California Verbal Learning Test (CVLT; Mulder *et al.*, 1996). In this task, a list of 16 items, with four words from each of four categories (fruit, herbs and spices, clothing, tools) is presented. Similar to the procedure in the AVLT, the list is presented five times, and the participant is instructed to recall as many words as possible from the list following each presentation. Verbal learning was assessed as the total number of recalled items on the five trials. The test-retest reliability of verbal learning in the Dutch CVLT has been examined in 17–74-year-old healthy subjects (Mulder *et al.*, 1996) and was .62 using a parallel test ( $n = 384$ ), and .58 when compared to the AVLT ( $n = 108$ ). In a pilot study of our twin family project we examined the test-retest reliability of the CVLT using a parallel test in 29 healthy adolescents (age 14–20 years) with an inter-test interval of 2–3 weeks (see Van Leeuwen, Van den Berg, Hoekstra & Boomsma, 2007, for details on the procedure of this pilot), and found a test-retest correlation of .86 for verbal learning.

In both the child and adolescent cohort, tests of verbal fluency were administered. This test evaluates the spontaneous production of words starting with a certain letter (verbal fluency letters) or belonging to a certain semantic category (verbal fluency categories) within a limited amount of time. The participants

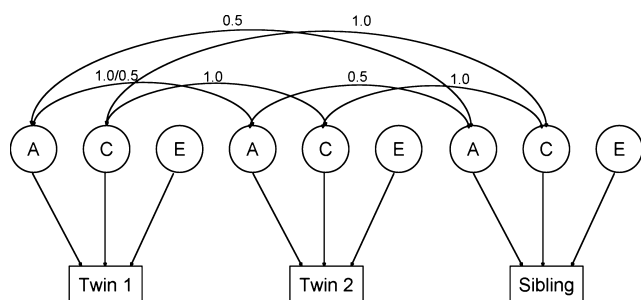
completed two trials for both conditions, and were instructed to name as many words as possible in 1 minute starting with an R or a T (letter trials), or belonging to the category 'animals' or 'professions' (category trials). Within the letter trials, participants were prohibited from saying proper nouns (e.g. Robert or Rotterdam) or saying the same word twice using a different ending (e.g. roast and roasted). To control for quantitative differences between trials within one condition (e.g. on average, the participants named more animals than professions), Z-scores were calculated for each trial. Letter fluency was measured as the mean Z-score over the two letter trials; category fluency was calculated as the mean Z-score of the semantic trials. In our pilot study a test-retest correlation of .70 for letter fluency and of .93 for category fluency was found.

### Statistical analyses

All analyses were carried out using structural equation modeling in the software package Mx (Neale, Boker, Xie & Maes, 2006). The significance of the effects of sex and age on the means of all verbal abilities was tested in a saturated model, which only specified that the multivariate data from family members could be correlated, but which did not impose any theoretical model on the covariance structure. The saturated model was also used to estimate the correlations between phenotypes within persons and the correlations between twins and between twins and siblings, both within and across traits (e.g. the correlation between VIQ in the oldest of the twins and verbal learning in the youngest of the twins). The covariance structure between family members and between traits was tested for equality across the age cohorts. All data were analyzed, including data from incomplete twin pairs and data from families without an additional sibling, using the raw data option in Mx.

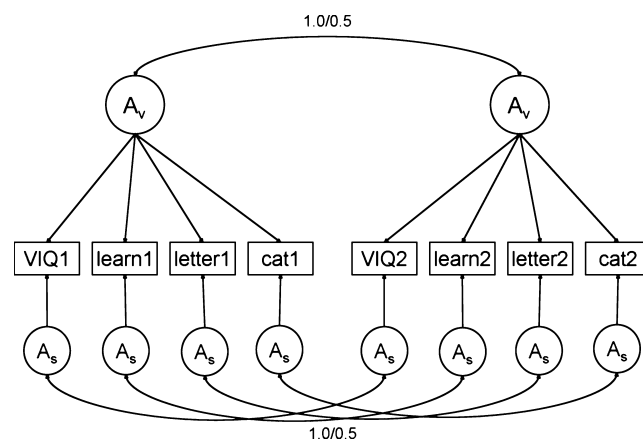
### Genetic modeling

Monozygotic twins are genetically identical at the DNA sequence level, while dizygotic twins and non-twin siblings share on average 50% of their segregating genes. Genetic model fitting of twin-sibling data allows one to attribute phenotypic variance and covariance into genetic and environmental components (Figure 1). Additive genetic influences (A) result from the additive effects of alleles at all contributing genetic loci. Shared environmental influences (C) represent the environmental effects common to all offspring from the same family. Nonshared environmental influences (E) are the effects of the environment that are not shared by the family members (including measurement error). Comparing the covariance structure of MZ pairs to that of DZ twins and twin-sibling pairs can give a first indication of what influences are important in explaining the (co)variance in test performance. If MZ and



**Figure 1** Univariate path diagram representing the contribution of additive genetic (A), shared environmental (C) and nonshared environmental (E) influences on the trait under investigation. The correlation of additive genetic factors is 1.0 in monozygotic twins, and, on average, 0.5 in dizygotic twins and between twins and siblings. The correlation of shared environmental effects is 1.0 between twins and between twins and siblings. Nonshared environmental effects represent influences unique to a family member and are thus uncorrelated.

first-degree relative within and across trait correlations are similar, shared environmental influences are likely to be important. Higher MZ correlations compared to DZ and twin–sibling correlations indicate that genetic effects play a role (Boomsma, Busjahn & Peltonen, 2002). By comparing the resemblance of DZ twins to the resemblance between twins and their non-twin siblings, it is possible to test whether there is evidence for a twin-specific environment. Higher DZ twin correlations compared to twin–sibling correlations would suggest such an effect. The relative importance of the components A, C, and E was estimated using structural equation modeling in Mx (Neale *et al.*, 2006). Genetic modeling was performed following several steps. The influences of A, C, and E on all verbal measures and on their overlap were first examined in a multivariate triangular or Cholesky decomposition (Neale & Cardon, 1992). A Cholesky decomposition yields the best possible fit to the data, as it is a fully parameterized model. The ACE Cholesky decomposition was applied separately to the data of the child cohort and the data of the adolescent cohort and was used to test whether the genetic and shared environmental effects were significant, by assessing the deterioration of the model fit after each component was dropped from the model. Next, we tested whether the genetic influences on all tests could be described by a genetic common factor model (Figure 2). This model assumes that there is one genetic factor plus test-specific genetic factors that influence the individual differences in each verbal test. A similar factor model was specified for the nonshared environmental influences. Next, a model was tested in which the nonshared environmental influences were constrained to be test-specific, but correlated between letter fluency and category fluency, as these variables were derived from the same type of test. A best fitting most parsimonious model was first established for the child



**Figure 2** Path diagram depicting a common factor model including test-specific influences. Path diagram shown for genetic effects, and for 2 family members only. This model can be expanded to include 3 family members, and can also be applied to environmental effects.  $A_v$  = Common genetic factor exerting its influence on all verbal abilities.  $A_s$  = test-specific genetic influences. Genetic effects correlate 1.0 between monozygotic twins, and on average 0.5 between dizygotic twins and twins and siblings. VIQ1/2 = verbal IQ family member 1/2; learn = verbal learning; letter = letter fluency; cat = category fluency.

cohort. Next, the genetic model fitting procedure was repeated for the data of the adolescent cohort.

The fit of the different submodels was evaluated against the saturated model using likelihood ratio tests and Akaike's information criterion. The likelihood ratio, which is the difference between minus twice the log likelihoods ( $-2 LL$ ) of two nested models, follows a  $\chi^2$  distribution. The degrees of freedom ( $df$ ) are given by the difference in the number of parameters estimated in the two models. A high increase in  $\chi^2$  against a low gain of degrees of freedom denotes a worse fit of the submodel compared to the full model. The most parsimonious model, with still a limited  $\chi^2$ , is chosen as the best fitting model. Some of the models (i.e. the common factor model for genetic vs. nonshared environmental influences) are not nested, and in these cases it is impossible to use the likelihood ratio test to evaluate which model fits better. In these instances, the Akaike's information criterion ( $AIC = \chi^2 - 2df$ ) was used. This fit statistic reflects the best balance between goodness of fit and parsimony with a model with the lowest AIC being the preferred model.

## Results

The descriptive statistics are given in Table 1 for the child and the adolescent cohorts. A positive age effect (i.e. better performance with increasing age) was found for all specific verbal measures. The age effect was not tested for VIQ, as this measure is standardized for age. Females outperformed males on verbal learning in both cohorts.

**Table 1** Descriptive statistics and the effects of age and sex for the verbal ability measures in 9-year-old twins and their siblings (child cohort), and in 18-year-old twins and their siblings (adolescent cohort)

	<i>n</i>	<i>M</i>	<i>SD</i>	Sex effect	Age effect
Child cohort					
VIQ	324	102.35	15.47	0.02	N/A
Verbal learning	323	38.40	7.44	3.16**	2.50**
Letter fluency	324	-0.39	0.77	0.29**	0.26**
Category fluency	324	-0.28	0.72	0.16	0.24**
Adolescent cohort					
VIQ	457	105.61	18.77	-1.20**	N/A
Verbal learning	456	56.35	7.99	2.73**	0.34*
Letter fluency	456	-0.096	0.87	0.15	0.09**
Category fluency	457	-0.02	0.84	0.00	0.10**

Note: \* =  $p < .05$ ; \*\* =  $p < .01$ . A negative sex effect denotes superior male performance; a positive effect represents better performance in females.

Girls performed better than boys on the letter fluency task in the child cohort, but this sex difference was not significant in the adolescent cohort. Males showed superior performance on the VIQ scale in the adolescent cohort. In the genetic analyses these effects on the means were included in the model (Neale *et al.*, 2006).

Differences in variances due to zygosity or twin-sibling status were absent within both cohorts. However, constraining the within-person variance/covariance structure to be equal across both cohorts resulted in a significant deterioration of the fit ( $\chi^2 = 42.19$ ,  $df = 10$ ,  $p < .001$ ). This indicates that the variance in the tests and the phenotypic covariance between the tests are significantly different in these two phases of development. The phenotypic correlations between measures are presented in Table 2, separately for the child (above diagonal) and the adolescent cohort (below diagonal). The difference in the multivariate structure between the two cohorts is mainly reflected in the significantly higher phenotypic correlation between VIQ and verbal learning and between letter and category fluency in the adolescent cohort. Overall, the phenotypic correlations are somewhat higher in the adolescent cohort (average  $r_{ph} = .40$ ) than in the child cohort (average  $r_{ph} = .30$ ).

Due to the different within-person variance/covariance structure in the two cohorts, subsequent analyses were

**Table 2** Phenotypic correlations (95% confidence intervals in parentheses) between verbal ability measures in the child cohort (above diagonal) and the adolescent cohort (below diagonal)

Task	VIQ	Verbal learning	Letter fluency	Category fluency
VIQ	—	.27 (.16–.38)	.35 (.24–.46)	.46 (.36–.54)
Verbal learning	.48 (.40–.55)	—	.24 (.13–.35)	.23 (.13–.34)
Letter fluency	.41 (.32–.49)	.29 (.20–.37)	—	.26 (.15–.36)
Category fluency	.39 (.30–.47)	.38 (.29–.46)	.47 (.39–.55)	—

performed separately for both age groups. Table 3 displays the correlations in MZ and DZ twins and between twins and their non-twin siblings for both the child (second figure on diagonal) and the adolescent cohort (first figure on diagonal). For all measures, MZ correlations are higher than DZ and twin-sibling correlations, indicating genetic influences. In the child cohort, the MZ correlations for VIQ and letter fluency are not twice as high as the DZ and twin-sibling correlations, suggesting that shared environmental influences may also play a role. In the adolescent cohort, the MZ cross correlations (off-diagonal of Table 3) are larger than the DZ and twin-sibling cross correlations. This pattern suggests that the overlap between various measures of verbal abilities is influenced by genetic effects. In the child cohort, most MZ cross correlations are higher than the DZ and twin-sibling correlations, indicating genetic influences. Constraining the covariance matrices to be equal in DZ twins and in twin-siblings did not result in a deterioration of the fit of the saturated model, either in the child ( $\chi^2 = 16.55$ ,  $df = 10$ ,  $p = .09$ ) or in the adolescent cohort ( $\chi^2 = 9.95$ ,  $df = 10$ ,  $p = .44$ ). Thus, there is no evidence for a twin-specific environment.

Table 4 gives the results of the model fitting for the saturated model and the more parsimonious sub-models. We started the model fitting procedure in the child cohort, by testing the significance of the shared environmental influences (model 2 in Table 4), and the additive genetic influences (model 3). Shared environmental effects were non-significant ( $\chi^2 = 2.03$ ,  $df = 10$ ,  $p = .99$ ). The additive genetic influences were significant ( $\chi^2 = 35.62$ ,  $df = 10$ ,  $p < .001$ ). Next it was tested whether the genetic effects on verbal abilities could be captured by a common factor model including test-specific effects (model 4). This model led to a significant drop in model fit ( $\chi^2 = 12.54$ ,  $df = 2$ ,  $p = .001$ ). A model in which the nonshared environmental influences were constrained to a common factor including test-specific effects (model 5) did fit the data well ( $\chi^2 = 1.67$ ,  $df = 2$ ,  $p = .43$ ). Lastly, a model with solely test-specific influences of the non-shared environment, but permitting covariance between letter fluency and category fluency (model 6), was fitted to the data of the child cohort. This model resulted in a significant deterioration of the fit ( $\chi^2 = 7.77$ ,  $df = 3$ ,  $p = .05$ ). All in all, the data of the child cohort were best described by a model including additive genetic and nonshared environmental effects, in which the nonshared environmental effects exert their influence through a common factor and test-specific influences (model 5). This model also had the lowest AIC value, indicating that it showed the best balance between parsimony and model fit. Subsequently, the model fitting procedure was repeated for the adolescent cohort data (bottom panel of Table 4). Similar to the results in the child cohort, the AE model with a common factor model including

**Table 3** Twin correlations and cross correlations (95% confidence intervals in parentheses) in MZ and DZ twins and between twins and siblings for all verbal ability tasks in two cohorts (child cohort above diagonal, adolescent cohort below diagonal)

Task	VIQ	Verbal learning	Letter fluency	Category fluency
<b>MZ</b>				
VIQ	<b>.84 (.78–.89)/ .82 (.73–.88)*</b>	.24 (.11–.37)	.34 (.20–.47)	.36 (.22–.47)
Verbal learning	.43 (.33–.52)	<b>.33 (.14–.49)/ .52 (.33–.66)*</b>	.30 (.13–.34)	.31 (.17–.45)
Letter fluency	.40 (.29–.49)	.27 (.13–.40)	<b>.51 (.33–.65)/ .37 (.07–.59)*</b>	–.03 (–.21–.15)
Category fluency	.33 (.23–.43)	.23 (.08–.35)	.37 (.23–.49)	<b>.55 (.37–.67)/ .46 (.22–.63)*</b>
<b>DZ</b>				
VIQ	<b>.33 (.16–.49)/ .66 (.49–.77)*</b>	.32 (.17–.46)	.25 (.09–.40)	.40 (.24–.53)
Verbal learning	.16 (.01–.30)	<b>.08 (–.13–.28)/ .13 (–.11–.34)*</b>	.16 (.00–.32)	.11 (–.07–.27)
Letter fluency	.17 (.04–.30)	.13 (.00–.26)	<b>.32 (.16–.47)/ .24 (.00–.44)*</b>	.12 (–.06–.28)
Category fluency	.10 (–.04–.23)	.10 (–.04–.24)	.11 (–.02–.24)	<b>.16 (–.02–.32)/ .27 (.01–.48)*</b>
<b>Twin–sibling</b>				
VIQ	<b>.41 (.26–.53)/ .45 (.30–.57)*</b>	.14 (.01–.26)	.21 (.09–.33)	.11 (–.02–.24)
Verbal learning	.25 (.13–.36)	<b>.15 (.00–.29)/ .20 (.04–.35)*</b>	.11 (.00–.22)	.06 (–.06–.17)
Letter fluency	.25 (.13–.36)	.11 (.00–.22)	<b>.36 (.21–.48)/ .24 (.10–.38)*</b>	.03 (–.08–.14)
Category fluency	.25 (.13–.35)	.05 (–.06–.16)	.22 (.11–.32)	<b>.21 (.07–.35)/ –.04 (–.20–.11)*</b>
<b>All 1st degree relatives</b>				
VIQ	<b>.38 (.26–.50)/ .48 (.35–.59)*</b>	.18 (.06–.29)	.22 (.11–.33)	.15 (.05–.27)
Verbal learning	.21 (.11–.31)	<b>.12 (.00–.25)/ .18 (.03–.32)*</b>	.12 (.02–.23)	.07 (–.03–.17)
Letter fluency	.22 (.12–.32)	.11 (.02–.21)	<b>.33 (.21–.44)/ .24 (.11–.37)*</b>	.05 (–.04–.15)
Category fluency	.18 (.08–.28)	.06 (–.03–.16)	.16 (.06–.26)	<b>.19 (.07–.31)/ .02 (–.11–.15)*</b>

Note: \* first correlation denotes resemblance in adolescent cohort, second correlation for child cohort.

**Table 4** Model fitting results for multivariate analyses of verbal abilities in the child and the adolescent cohort

Model	df	–2LL	cpm	$\chi^2$	$\Delta df$	p	AIC
<b>Child cohort</b>							
1. ACE Cholesky	1256	6056.64					
2. AE Cholesky	1266	6058.67	1	2.03	10	.99	–17.97
3. CE Cholesky	1266	6092.26	1	35.62	10	<.001	15.62
4. AE A common factor + test-specific	1268	6071.21	2	12.54	2	.001	8.54
5. AE E common factor + test-specific	1268	6060.34	2	1.67	2	.43	–2.33
			1	3.70	12	.99	–20.30
6. AE E test-specific + correlated E between verbal fluency tests	1271	6068.11	5	7.77	3	.05	1.77
<b>Adolescent cohort</b>							
1. ACE Cholesky	1787	8914.56					
2. AE Cholesky	1797	8917.03	1	2.47	10	.99	–17.53
3. CE Cholesky	1797	8970.80	1	56.23	10	<.001	36.23
4. AE A common factor + test-specific	1799	8926.05	2	9.02	2	.01	5.02
5. AE E common factor + test-specific	1799	8917.57	2	.54	2	.76	–3.46
			1	3.01	12	.99	–20.99
6. AE E test-specific + correlated E between verbal fluency tests	1802	8934.39	5	16.82	3	<.001	10.82

Note: –2LL = –2 log likelihood; df = degrees of freedom; cpm = compared to model.

test-specific effects describing the influences of the nonshared environment fit the data best (model 5,  $\chi^2 = .54$ ,  $df = 2$ ,  $p = .76$ , AIC = –3.46).

The relative importance of additive genetic and nonshared environmental effects on the variance in each test is given on the diagonal in Table 5 (child



**Table 5** Contributions of additive genetic (A) and nonshared environmental (E) effects to the variance and covariance in verbal abilities, and the genetic correlations ( $r_g$ ) between these abilities, in the child cohort. Estimates are based on the best fitting model (95% confidence interval in parentheses)

	VIQ	Verbal learning	Letter fluency	Category fluency	Estimates corrected for measurement error
<b>A</b>					
VIQ	<b>.81 (.72–.88)</b>				A corrected
Verbal learning	1.00 (.89–1.00)	<b>.46 (.29–.62)</b>			.97
Letter fluency	.88 (.71–.97)	1.00 (.80–1.00)	<b>.40 (.23–.57)</b>		.95
Category fluency	.72 (.46–.92)	1.00 (.51–1.00)	.11 (.00–.64)	<b>.29 (.13–.50)</b>	.82
<b>E</b>					
VIQ	<b>.19 (.12–.28)</b>				E corrected
Verbal learning	.00 (.00–.11)	<b>.54 (.38–.71)</b>			.03
Letter fluency	.12 (.03–.29)	.00 (.00–.20)	<b>.60 (.43–.77)</b>		.05
Category fluency	.28 (.08–.54)	.00 (.00–.49)	.89 (.36–1.00)	<b>.71 (.50–.87)</b>	.18
<b><math>r_g</math></b>					
VIQ	–				
Verbal learning	.42 (.23–.60)	–			
Letter fluency	.53 (.33–.74)	.55 (.30–.78)	–		
Category fluency	.67 (.43–.89)	.70 (.33–.95)	.09 (.00–.53)	–	

**Table 6** Contributions of additive genetic (A) and nonshared environmental (E) effects to the variance and covariance in verbal abilities, and the genetic correlations ( $r_g$ ) between these abilities, in the adolescent cohort. Estimates are based on the best fitting model (95% confidence interval in parentheses)

	VIQ	Verbal learning	Letter fluency	Category fluency	Estimates corrected for measurement error
<b>A</b>					
VIQ	<b>.84 (.78–.89)</b>				A corrected
Verbal learning	.96 (.82–1.00)	<b>.29 (.17–.43)</b>			.99
Letter fluency	.97 (.87–1.00)	.81 (.46–.95)	<b>.55 (.42–.66)</b>		.76
Category fluency	.88 (.68–1.00)	.44 (.12–.73)	.73 (.50–.92)	<b>.47 (.31–.61)</b>	1.00
<b>E</b>					
VIQ	<b>.16 (.11–.22)</b>				E corrected
Verbal learning	.04 (.00–.18)	<b>.71 (.57–.83)</b>			.01
Letter fluency	.03 (.00–.13)	.19 (.05–.54)	<b>.45 (.34–.58)</b>		.24
Category fluency	.12 (.00–.32)	.56 (.27–.88)	.27 (.08–.50)	<b>.53 (.39–.69)</b>	.00
<b><math>r_g</math></b>					
VIQ	–				
Verbal learning	.92 (.73–1.00)	–			
Letter fluency	.59 (.46–.70)	.58 (.32–.80)	–		
Category fluency	.54 (.37–.71)	.45 (.14–.71)	.67 (.48–.84)	–	

cohort) and Table 6 (adolescent cohort). In both age groups, the heritability was strongest for VIQ. Against expectations, the point estimate for heritability of verbal learning was stronger in the child than in the adolescent cohort, although the confidence intervals overlap. Following our expectations, the point estimates for the heritability of letter and category fluency were higher in the adolescent cohort. The estimates of the nonshared environmental influences also include measurement error. When measurement error is taken into account (based on the test–retest reliability statistics of each measure) it is revealed that in both cohorts most of the reliable trait variance in VIQ, verbal learning and letter fluency was explained by genetic effects (see the far right panel of Tables 5 and 6). Category fluency, however, showed substantial nonshared environmental influences other than measurement error.

The contributions of genes and environment on the covariance between the different verbal abilities are given

on the subdiagonals of Tables 5 and 6. In both cohorts, genetic effects account for most of the overlap between the tests. The only exception is the covariance between letter and category fluency in the child cohort, on which genetic effects are not significant. In the adolescent cohort, the nonshared environmental influences are substantial on the covariance between verbal learning and category fluency. It should be noted that the confidence intervals around these estimates are large. The genetic correlations between the different tests are given at the bottom of Tables 5 and 6. On the whole, the genetic correlations are stronger in the adolescent cohort (average  $r_g$  over all tests = .63) compared to the child cohort (average  $r_g$  = .49). The genetic correlations in the adolescent cohort range from .45 to .92, with the genetic correlation between VIQ and verbal learning not being significantly different from unity. In the child cohort the genetic correlations range between .42 and .70, with one notable exception. The genetic correlation between letter

and category fluency is only modest and not significantly different from zero. These results suggest that the higher phenotypic correlations in the adolescent cohort compared to the child cohort can be explained by stronger correlations between the tests on the genetic level.

## Discussion

We studied the etiology of the overlap in VIQ, verbal learning, letter and category fluency in middle childhood and late adolescence. Both cohorts included twins and non-twin siblings and showed no evidence of a twin-specific environment. In both cohorts, the individual differences in VIQ were strongly influenced by genetic effects (84% and 82%), while the performance in more specific lexicon-related abilities was under moderate genetic influence (29–55%). The remaining variance was explained by nonshared environmental effects including measurement error. Genetic effects were of major importance in explaining the overlap between the different verbal abilities, in both the child and the adolescent cohorts. A common factor structure exerting its influence on all tests accounted for the nonshared environmental covariance between the tests. The main difference between the two cohorts lay in the stronger correlations between some of the verbal tests in the adolescent cohort at the genetic level. The higher genetic correlations resulted in a stronger phenotypic overlap between some of the verbal tests in the adolescent cohort. These results and their implications are discussed in more detail below.

### *Phenotypic correlations between verbal tasks in middle childhood and late adolescence*

The within-person variance and covariance structure between the tests was found to be different in middle childhood and late adolescence. The phenotypic correlations between VIQ and verbal learning and between letter and category fluency were significantly higher in the adolescent cohort. The higher phenotypic correlations in the adolescent sample were not so much due to stronger genetic influences on each individual test, but rather to increased genetic correlations between some of the tests. These results suggest that verbal abilities may be more generalized at a later stage of development. It should be noted that verbal learning was assessed using slightly different tests in the two cohorts (the AVLTL in the child cohort vs. the CVLT in the adolescent cohort). Although previous studies indicated strong overlap between AVLTL and CVLT performance (Mulder *et al.*, 1996; Stallings, Boake & Sherer, 1995), with correlations close to the test–retest correlations of the CVLT itself (Mulder *et al.*, 1996), we cannot exclude the possibility that these differences have affected the pattern of phenotypic correlations.

The phenotypic correlations between verbal learning, letter and category fluency were moderate in both cohorts. The phenotypic correlations between letter fluency and category fluency were relatively low ( $r = .26$  in the child cohort and  $.47$  in the adolescent cohort), given that the same type of test was used. This finding suggests that these tasks tap different aspects of word fluency. Letter fluency requires phoneme analysis, while category fluency relies more heavily on semantic memory. Category fluency performance is shown to be superior in children who frequently use schemata to guide their recall (Sincoff & Sternberg, 1988). For instance, the participants in our study could improve their performance on the ‘animal’ trial of category fluency by thinking of all the animals that live in a zoo or on a farm. It is also possible to follow strategies in a letter fluency task (e.g. name words starting with the same consonants, such as *reptile* and *replication*), but these strategies are not as obvious, and less often used.

### *Heritability of general vs. specific verbal abilities*

Individual differences in general verbal abilities, as measured with the Wechsler VIQ, were found to be highly heritable, both in middle childhood and in late adolescence. The heritability estimate of 84% found in our sample of 18-year-old twins and their siblings is similar to heritability estimates of VIQ in other adult samples, that reported a heritability of 84% (Rijsdijk *et al.*, 2002) and 85% (Posthuma *et al.*, 2001). The heritability estimate of 82% for VIQ in 9-year-old twins and their siblings is somewhat higher than the estimates reported in other studies in middle childhood. Hoekstra *et al.* (2007) reported a modest influence of shared environmental influences (16%) in 10-year-old twins. Based on a comparison of the MZ and first-degree relatives correlations in our current child data ( $r = .82$  and  $r = .48$ , respectively), a modest effect of the shared environment would be expected (because the correlation in first-degree relatives is higher than half the MZ correlation). Regrettably the sample size of our current study ( $n = 324$ ) gives insufficient power to detect these modest effects. We conducted a power analysis and found that a sample size of nearly 700 children would be needed to have sufficient power to detect these modest influences of the shared environment. In the adolescent sample, the MZ correlation was more than twice as high as the correlation between first-degree relatives, yielding no indication for an influence of shared environment on VIQ.

The genetic influences on the variance in verbal learning were moderate in both cohorts. Against expectations, the point estimate of the genetic effects was higher in middle childhood (46%) than in late adolescence (29%), although the confidence intervals overlap. The attenuated genetic effects in the adolescent cohort compared to the child cohort are most likely explained by differences in the tests used to measure

these abilities. In the child cohort, verbal learning was assessed with the AVLTL, in which a list of unrelated words is used. In the adolescent cohort, learning and memory performance was determined with the CVLT, including a list of words belonging to different categories. One previous twin study examined the heritability of uncategorized word learning versus categorized word learning (Volk *et al.*, 2006), and found stronger genetic effects on uncategorized (55%) than on categorized learning (38%). This difference in heritability could explain why the heritability estimate for learning was higher in the child cohort compared to the adolescent cohort. Genetic effects on letter and category fluency were moderate in the child cohort (40% and 29%, respectively). Although confidence intervals overlap, the point estimates for the genetic effects on both measures were somewhat higher in the adolescent cohort (55% and 47%), consistent with the increasing genetic influences on verbal abilities found in previous studies (Alarcón *et al.*, 2003; Alarcón *et al.*, 1998; Alarcón *et al.*, 1999; Hoekstra *et al.*, 2007).

The remaining variance in all tasks was explained by nonshared environmental influences. These influences also include measurement error. Partialling out these effects showed that only category fluency was influenced by substantial nonshared environmental effects other than scale unreliability. Shared environmental influences failed to be significant, both in the child and the adolescent cohort. The lack of shared environmental influences on individual differences in adult verbal abilities is in accordance with findings from previous studies (Ando *et al.*, 2001; Posthuma *et al.*, 2001; Rijdsdijk *et al.*, 2002; Swan & Carmelli, 2002; Swan *et al.*, 1999; Volk *et al.*, 2006). Studies in early to middle childhood on verbal fluency and verbal memory reported modest (Samuelsson *et al.*, 2005) or non-significant effects of the shared environment (Bishop *et al.*, 2006; Kovas *et al.*, 2005; Thompson *et al.*, 1991). The results of the current study do not provide evidence for a strong influence of the shared environment on verbal learning and fluency in middle childhood and adolescence. However, similar to the findings of VIQ in middle childhood, a modest influence of the shared environment cannot be excluded, due to power restrictions to detect these influences.

#### *Genetic and environmental covariation between different verbal tests*

Genetic influences appeared to be the driving force behind the covariation between verbal abilities. The association between VIQ and the specific verbal tasks was almost entirely explained by genetic effects in both cohorts. The overlap between verbal learning, letter and category fluency was explained by both genetic effects and nonshared environmental influences, but these influences should be interpreted with care as the confidence intervals around the estimates vary widely.

The nonshared environmental effects on the covariance between the verbal tests were best described by a common factor model. This finding implies that there is one nonshared environmental factor, albeit of moderate impact, that influences the performance of all verbal tests. Possible nonshared environmental effects on verbal abilities could include traumatic experiences not shared with the other family members, or consequences of an accident or illness. Perinatal factors such as low birth weight and intrapartum complications may affect language development (Stromswold, 2006). These factors are not necessarily the same for both members of a twin pair and might therefore be reflected in the nonshared environmental effects. Also, if the children are in separate classes, the influences of the teacher or other school-related influences will be nonshared. Furthermore, child-specific influences, such as weariness on the day of testing, may account for the covariance between tests.

With one notable exception in the child cohort (between letter and category fluency), all genetic correlations between the different verbal measures are substantial, both in the child and in the adolescent cohort. This finding indicates that general verbal ability and more specific measures linked to the mental lexicon are largely influenced by the same set of genes. These findings are similar to earlier studies in early childhood (Dale *et al.*, 2000; Hayiou-Thomas, Kovas, Harlaar, Plomin, Bishop & Dale, 2006) and adulthood (Ando *et al.*, 2001) that reported common genetic influences on various verbal abilities. Dale *et al.* (2000) found that measures of vocabulary and grammar were substantially correlated in 2-year-old twins, both at the phenotypic ( $r_{ph} = .66$ ) and the genetic ( $r_g = .61$ ) level. In 4.5-year-old twins from TEDS, verbal category fluency correlated moderately with other measures of language development, and the genetic correlations were substantial, ranging from .48 to .96 (Hayiou-Thomas *et al.*, 2006). In Ando *et al.*'s (2001) study of adult twins, a common genetic factor explained 20–26% of the variance in the verbal tasks, suggesting that some of the genetic influences were general, while the rest of the variance was modality or test-specific. In our study, although the genetic correlations were high, a genetic common factor model could not be fitted to the data without a significant reduction of the model fit, either in the child or in the adolescent cohort. This finding indicates that verbal abilities are not entirely unidimensional on the genetic level.

#### *Implications for future studies*

The results from this study are also relevant to research in psychopathology. Several clinical studies have reported impaired performance on verbal learning as measured with the CVLT or the AVLTL in patients suffering from schizophrenia (Appels *et al.*, 2003; Egan *et al.*, 2001; Simon *et al.*, 2007; Weickert *et al.*, 2000), or their

relatives (Appels *et al.*, 2003; Egan *et al.*, 2001; Snitz, Macdonald & Carter, 2006; Szöke, Schurhoff, Mathieu, Meary, Ionescu & Leboyer, 2005). Moreover, some studies reported impaired performance on letter or category fluency in schizophrenia patients and their relatives (Appels *et al.*, 2003; Chen *et al.*, 2000; Snitz *et al.*, 2006; Szöke *et al.*, 2005), and in (relatives of) children diagnosed with an autism spectrum disorder (Geurts *et al.*, 2004; Hughes, Plumet & Leboyer, 1999). Following this, measures of verbal learning and fluency have been proposed as promising endophenotypes for psychiatric illness. One of the criteria for a good endophenotype is that the endophenotype itself should be under substantial genetic control (De Geus & Boomsma, 2001; Viding & Blakemore, 2007). The current study provides a direct test of this criterion. The results of our study, together with the findings of Volk *et al.* (2006), suggest that individual differences in verbal learning as measured with the AVLTL are more strongly genetically determined than as measured with the CVLT. Therefore, we suggest the use of the AVLTL if researchers plan to use verbal learning as an endophenotype.

Both letter fluency and category fluency are under moderate genetic influence, and both therefore could serve as useful endophenotypes. However, it is important to note that the genetic correlation between these measures is relatively low (and not significantly different from zero in the child cohort). Researchers should probably avoid including a composite score of 'overall verbal fluency' as an endophenotype, as these measures reflect genetically different cognitive constructs. Consistent with this finding are the results from imaging studies, which suggest that these tasks are sensitive to partly different neuroanatomical substrates (Costafreda, Fu, Lee, Everitt, Brammer & David, 2006; Heim, Eickhoff & Amunts, 2008).

Lastly, VIQ was under stronger genetic influence than the more specialized verbal abilities in both age groups. Moreover, the correlations between VIQ and the more specific verbal tasks were only moderate. Researchers interested in the genetic effects on general verbal abilities should therefore be aware that these abilities are not simply captured by a quick and easy to administer test such as verbal fluency.

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